opacities similar to those seen in silicosis.[973, 977] Spontaneous pneumothorax occurs in slightly more than 10% of cases.[971] Rare complications include fungus ball formation[978] and extensive calcification of the nodules.[977]

Clinical Manifestations

The majority of patients reported to have chronic beryllosis have been exposed to the dust for more than 2 years. Typically, symptoms develop insidiously after a latent period that may be as long as 15 years after the last exposure to dust.[948, 979, 980] Occasionally, patients have minimal symptoms and radiographic manifestations.[883] Some investigators believe that the disease can be precipitated by certain trigger factors, such as pregnancy, withdrawal from exposure, and even the performance of a beryllium patch test.[950, 981]

Early symptoms include cough, fatigue, weight loss, increasing dyspnea on exertion, and, sometimes, migratory arthralgia. Crackles may be heard on auscultation, and the liver and spleen may be palpable. With progression of disease, cyanosis may become evident, and in approximately 30% of patients, clubbing of the fingers and toes develops; cor pulmonale is frequent at this stage. Symptoms of systemic disease may be related to myocarditis, gout, and neprolithiasis. Dermal lesions similar to those seen in sarcoidosis may occur.

Laboratory Findings and Diagnosis

Hypergammaglobulinemia, hypercalciuria, hyperuricemia, and polycythemia are not uncommon findings;[948, 979, 980, 982] up to 10% of patients develop renal calculi. The serum angiotensin-converting enzyme level may be elevated, although this may also be seen in healthy beryllium-exposed workers.[983] The total number of cells obtained by BAL is increased, principally owing to larger numbers of lymphocytes;[954] most are CD4+ T cells,[957] and there is consequently an increased helper-to-suppressor ratio. In nonsmokers, the intensity of this "lymphocytic alveolitis" correlates with the clinical severity of the berylliosis.[984]

Confirmation of the diagnosis may be obtained by a patch test showing hypersensitivity to beryllium.[985, 986] The proliferative response to beryllium of lymphocytes in specimens obtained by BAL has also been proposed as a useful diagnostic test,[941, 987–989] although its expense and time-consuming nature may preclude its use as a screening tool.[990] A positive test may precede the development of clinically evident disease or abnormalities of pulmonary function.[991, 992] Abnormal peripheral blood lymphocyte proliferation on exposure to beryllium is seen in most patients;[951, 955, 991] this test may be useful in differential diagnosis of other granulomatous disorders[989] because beryllium-exposed workers who do not have disease or sensitization do not demonstrate this response. In one study of a small number of patients who had chronic berylliosis, one group of workers found this test to show 100% sensitivity and specificity.[988] In one study, a serum neopterin value of 1.27 ng/ml in workers who had an abnormal beryllium lymphocyte proliferation test was 88% specific and had a positive predictive value of 92% for the diagnosis of chronic beryllium disease.[993]

For obvious reasons, the disease may be confused with sarcoidosis.[941, 994] Careful elucidation of any history of possible exposure to beryllium helps prevent diagnostic error. As indicated previously, although there is overlap between healthy individuals and patients who have sarcoidosis or berylliosis, the tissue level of beryllium is generally higher in berylliosis, and its measurement may be helpful in some cases in which exposure history is unclear.[968]

Pulmonary Function Tests

Abnormalities of pulmonary function are common.[996] In one investigation of 41 patients, they were apparent in 39;[997] 16 manifested an obstructive pattern, 8 manifested a restrictive one, and 15 had diminished diffusing capacity without evidence of either obstruction or restriction. Patients who had obstructive lung disease were not necessarily smokers. Similar findings were reported in a more recent study.[998] Evidence of functional derangement may precede radiographic evidence of disease;[999] the first measurable abnormality is likely an elevation of dead space ventilation on exercise.[998] In more advanced disease, Pa_{O_2} is decreased, even at rest. Diffusing capacity may be reduced, and alveolar-arterial oxygen difference may be increased. A few patients have functional impairment suggestive of emphysema.

Prognosis and Natural History

The prognosis of patients who have symptomatic, chronic berylliosis is poor,[997, 998] particularly when there is complicating cor pulmonale. There is evidence that the presence of a granulomatous reaction in the lung parenchyma as opposed to simple mononuclear inflammatory cell infiltration is associated with a better prognosis.[945] Despite these findings, there is evidence that a reduction in the concentration of beryllium in the air can result in a significant improvement in lung function. For example, in one study of 20 men who had hypoxemia at the time air pollution was reduced, 13 showed improvement in arterial blood gases and lower alveolar-arterial gradients in a follow-up study 3 years later;[1000] some of these patients also showed an improvement in the severity of radiographic abnormalities.

Beryllium is a well-known carcinogen that is capable of causing pulmonary carcinoma in animals.[1001] Large cohort studies support the hypothesis that it is a carcinogen for humans as well,[1002, 1003] particularly for those individuals who have suffered from acute berylliosis.

Aluminum

Because of its versatility and metallic properties, occupational contact with aluminum is common: in the United States, approximately 2 million individuals are exposed directly to aluminum oxide, and almost the same number have contact with base or coated metal.[1004] Exposure can occur in several situations, including the following:

1. In the reduction of alumina to metallic aluminum during the process of smelting.[1005] This occurs in large rooms (pot rooms) that contain many potentially toxic gases and fumes in addition to aluminum dust. Although bauxite itself is generally believed to be innocuous, the results of one pathologic study suggested that it may cause pulmonary

fibrosis.[1007] In addition, workers involved in the mining and refining of bauxite have been found to have an increasing prevalence of low-profusion reticular densities radiologically with increasing dust exposure, an effect enhanced by smoking but unexplained by age.[1008]

2. During the preparation or use of aluminum powder derived either from stamping of cold metal (flake type) or directly from molten metal (granular type).[1009–1013]

3. During aluminum arc welding.[1014–1016]

4. During the grinding or polishing of aluminum products[1017, 1018] or in the manufacture or use of aluminum-based abrasive grinding tools.[1004]

Although each of these situations has been associated with pulmonary disease, it is not certain that aluminum is the pathogenetic agent in every instance because there is often concomitant exposure to other potentially toxic substances.[1005, 1019] This pathogenetic uncertainty is supported by experimental animal studies in which there has been minimal or no pulmonary reaction to inhaled aluminum.[1020, 1021] In addition, after the observation that silicosis does not develop in rabbits that inhale dust containing 1 part of aluminum to 100 parts of freshly fractured quartz,[1022] aluminum was added prophylactically to dust inhaled by miners exposed to silica; such addition apparently has had no untoward effects.[1023] Despite these findings, other animal experiments have been associated with a significant fibrotic reaction to inhaled aluminum,[1024, 1025] implying that true toxicity may occur in some situations. Although differences in the species, method of aluminum administration, or form of aluminum employed (e.g., fibrous versus nonfibrous[1004]) may underlie the discrepant findings in the experimental studies, it has been hypothesized that host factors, perhaps mediated by immunologic mechanisms, may also be responsible.[1013, 1016]

Pathologic findings in the lungs of individuals exposed to aluminum are variable and, as indicated, may be caused in some cases by substances other than aluminum itself. Diffuse interstitial fibrosis has been described in workers engaged in smelting[1026, 1027] and in the production of grinding wheels[1004] and abrasives.[1010] Other histologic reactions that have been reported include desquamative interstitial pneumonitis,[1014] alveolar proteinosis,[1017] and diffuse granulomatous inflammation.[1013, 1016]

Radiographic abnormalities may become apparent after a few months or several years of exposure.[215, 1011] Fully developed changes consist of a fine-to-coarse reticular pattern widely distributed throughout the lungs (Fig. 60–61), sometimes with a nodular component.[1011] The fibrosis fre-

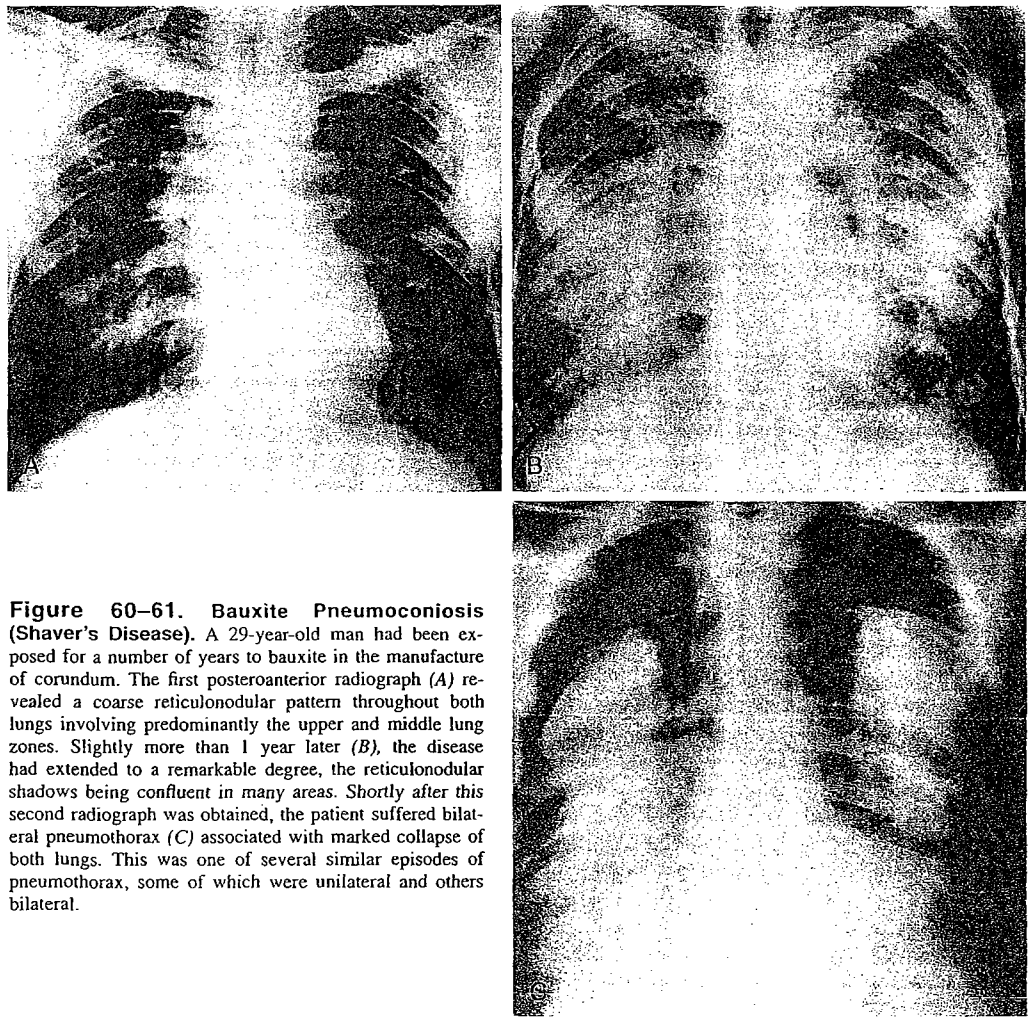

Figure 60–61. Bauxite Pneumoconiosis (Shaver's Disease). A 29-year-old man had been exposed for a number of years to bauxite in the manufacture of corundum. The first posteroanterior radiograph *(A)* revealed a coarse reticulonodular pattern throughout both lungs involving predominantly the upper and middle lung zones. Slightly more than 1 year later *(B)*, the disease had extended to a remarkable degree, the reticulonodular shadows being confluent in many areas. Shortly after this second radiograph was obtained, the patient suffered bilateral pneumothorax *(C)* associated with marked collapse of both lungs. This was one of several similar episodes of pneumothorax, some of which were unilateral and others bilateral.

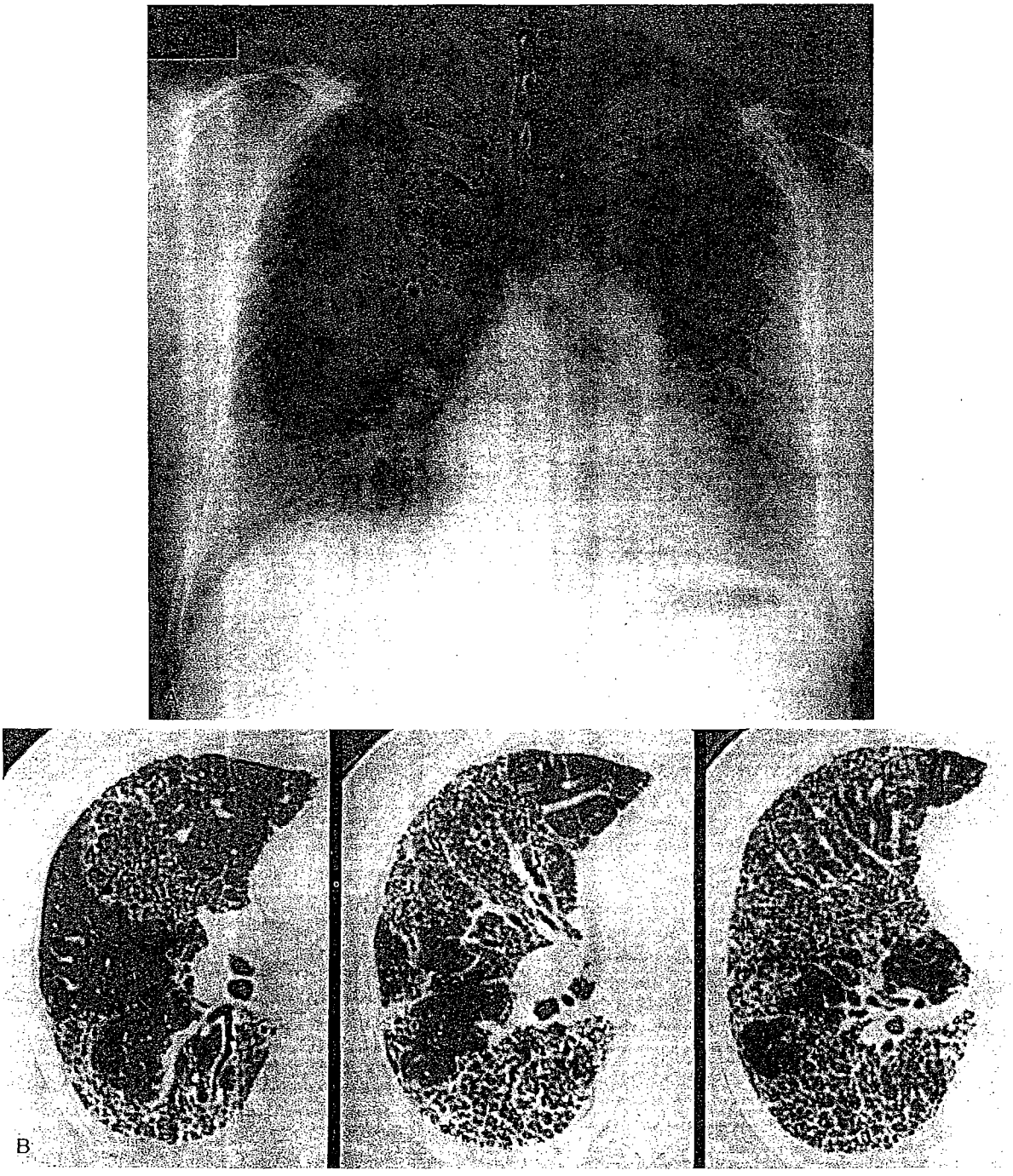

Figure 60–62. Aluminum Pneumoconiosis. A 52-year-old man with a history of exposure to aluminum for 7 years presented with exertional dyspnea. A posteroanterior chest radiograph *(A)* demonstrates a diffuse, bilateral reticular pattern. HRCT images targeted to the right lung *(B)* better demonstrate the reticular pattern and the presence of honeycombing. (Courtesy of Dr. Masanori Akira, Department of Radiology, National Kinki Chuo Hospital Chest Disease, Osaka, Japan.)

quently involves the upper lobes.[1004] HRCT findings have been described in one study of six workers, in whom the abnormalities consisted of predominantly small nodular opacities in two and a reticular pattern in four;[1029] honeycombing was also present in two patients (Fig. 60–62). In five of the six patients, the abnormalities involved mainly the upper lung zones. Lung volume may be greatly decreased, and the pleura may become thickened; spontaneous pneumothorax is a frequent complication (*see* Fig. 60–61).

Breathlessness is the chief symptom; in severe cases, it may be disabling and lead to death from pulmonary insufficiency.[1010, 1011] Work in aluminum smelters has been

associated with the development of both asthma and chronic air-flow obstruction.[1030–1039] Pulmonary function studies have shown both restrictive and obstructive disease with reduction in diffusing capacity.[215, 1004] No association with the development of carcinoma was found in one cohort study.[1006]

Cobalt and Tungsten Carbide

The term *hard metal* is usually used to refer to an alloy of tungsten, carbon, and cobalt, occasionally with the addition of small amounts of other metals, such as titanium, tantalum, nickel, and chromium.[1040, 1041] The resulting product is extremely hard and resistant to heat and is used extensively in the drilling and polishing of other metals. Exposure to dust can occur during either the manufacture or the use of the metal and is well recognized as a cause of interstitial pneumonitis and fibrosis.[1042–1046]

The etiology and pathogenesis of disease are unclear. The results of experimental studies in animals suggest that cobalt is the causative agent,[1041, 1047] a hypothesis supported by the observation that diamond polishers—who are exposed to high concentrations of cobalt alone—develop pulmonary disease virtually identical to that seen in hard metal workers.[1048] There is evidence, however, that the effects of cobalt are enhanced by the presence of tungsten carbide;[1041] moreover, in some autopsy studies of patients who have interstitial fibrosis and a history of exposure to hard metals, cobalt has not been found in the lung tissue.[1049, 1050] It has thus been suggested that the disease may result in some workers from a hypersensitivity reaction analogous to that seen in berylliosis.[1044, 1049] Some cases of asthma have also been associated with cobalt exposure;[1051, 1052] there is evidence that this may be caused by an immunologically mediated hypersensitivity reaction,[1053, 1054] possibly enhanced by cigarette smoking.[1055]

Pathologic findings are predominantly those of interstitial pneumonitis and fibrosis.[1040, 1047] Characteristically, numerous macrophages are present in alveolar air spaces, creating a pattern simulating desquamative interstitial pneumonitis. In many cases, multinucleated giant cells are prominent, both in the air spaces and lining alveolar walls, resulting in a pattern of giant cell interstitial pneumonitis (Fig. 60–63);[1040, 1044, 1056, 1057] the giant cells can also be seen in cytology specimens obtained by bronchial washing.[1058] Obliterative bronchiolitis has been noted occasionally.[1040, 1056] Particulate material may or may not be identified within the macrophages or giant cells by light microscopy; spectroscopic analysis reveals predominantly tungsten with little evidence of cobalt.[1040]

The radiographic findings consist of a diffuse micronodular and reticular pattern, sometimes associated with lymph node enlargement; the reticulation may be coarse[1059] and in advanced disease may be accompanied by small cystic spaces.[1047, 1060] In one study of two hard metal workers, the HRCT findings consisted of bilateral areas of ground-glass attenuation, areas of consolidation, and extensive reticular opacities and traction bronchiectasis indicative of fibrosis (Fig. 60–64);[1029] autopsy correlation in one case showed the areas of ground-glass attenuation and consolidation to correspond to aggregates of mononuclear and multinucleated giant cells.

Symptoms include cough, sometimes productive,[1043]

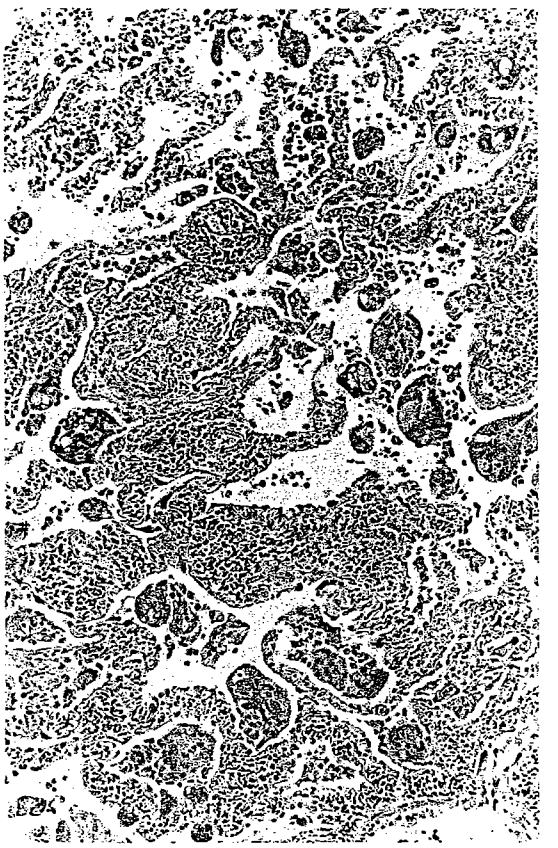

Figure 60–63. Hard Metal Lung Disease. The section shows moderately severe interstitial pneumonitis and fibrosis and the presence of a large number of irregular multinucleated giant cells in the alveolar air spaces. (×80.)

and dyspnea on exertion; severe respiratory insufficiency sometimes develops and can prove fatal.[1059, 1061, 1062] Weight loss out of proportion to the degree of respiratory impairment is frequently seen, possibly related to the elaboration of TNF-α by lung inflammatory cells.[1063]

In the appropriate clinical setting, the identification of multinucleated giant cells in BAL fluid supports the diagnosis. Eosinophilia was noted in the BAL fluid of one worker who had combined heavy metal and aluminum dust exposure.[1064] Pulmonary function tests reveal both restrictive[1043, 1065] and obstructive patterns,[1066] and diffusing capacity may be reduced.[1043, 1047, 1065] Minor alterations in spirometry without radiologic abnormalities were described in a group of diamond polishers[1066a] and in a group of saw filers[1067] exposed to "high" cobalt exposure, which nevertheless respected the industry threshold limit value for cobalt.

Silicon Carbide

Silicon carbide (carborundum) is produced by fusion at high temperature of high-grade sand, finely ground carbon (coke), salt, and wood dust.[1068] The resulting product is extremely hard and is used as an abrasive. Although the findings of experimental animal studies have suggested that

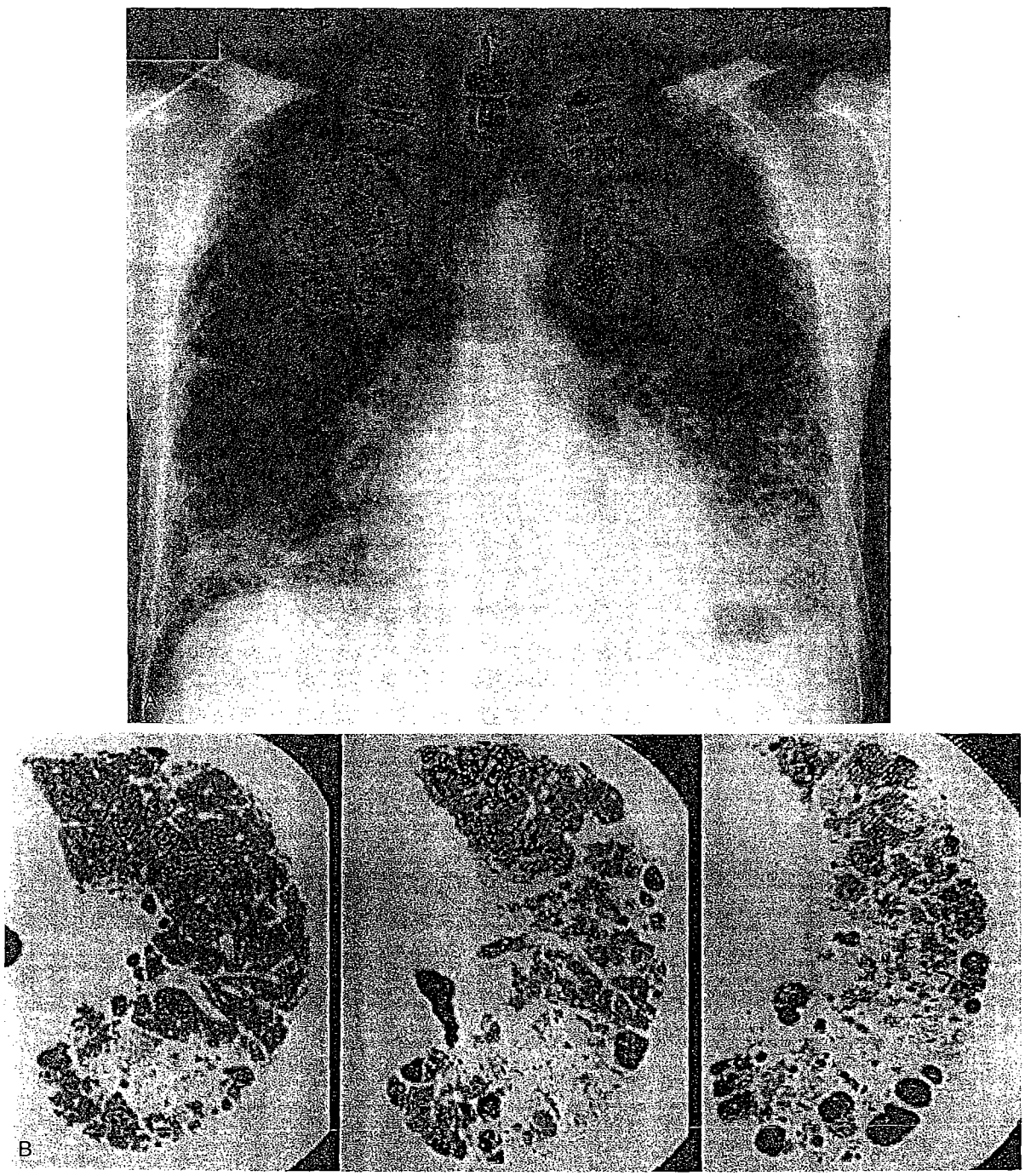

Figure 60–64. Hard Metal Lung Disease. A 45-year-old Japanese man presented with exertional dyspnea. He had a history of exposure to hard metal for 5 years. A chest radiograph *(A)* demonstrates a coarse reticular pattern involving mainly the peripheral lung regions and the lower lung zones. HRCT images targeted to the left lung *(B)* demonstrate extensive areas of ground-glass attenuation. Irregular linear opacities and traction bronchiectasis consistent with fibrosis are also evident. Several cystic spaces consistent with end-stage honeycombing are present in the subpleural lung regions. (Courtesy of Dr. Masanori Akira, Department of Radiology, National Kinki Chuo Hospital Chest Disease, Osaka, Japan.)

the substance is inert,[1068–1070] workers in the carborundum industry have had pathologic evidence of interstitial fibrosis and macrophage accumulation[1071] accompanied by radiographic and pulmonary function abnormalities.[1069, 1072–1075] It is not certain to what extent these changes are caused by silica derived from the sand, by other contaminants in the dust such as cristobalite or tridymite, or by silicon carbide fibers produced during the manufacturing process.[1069, 1076, 1077]

Silicon carbide can be identified in tissue sections as thin black fibers often associated with an iron-protein coat (ferruginous bodies) (Fig. 60–65). Some investigators have suggested a possible association of exposure with pulmonary carcinoma;[1071] however, in one large epidemiologic study, no evidence of excess deaths from this cause was found.[1078] Radiographic findings include nodular, reticulonodular, or reticular opacities with or without hilar lymphadenopathy (Fig. 60–66).[1079, 1081] Pleural plaques similar to those seen in asbestos-exposed individuals have also been described.[1075]

Polyvinyl Chloride

In its pure form, polyvinyl chloride is a white powder that is produced by polymerization under pressure of the gas vinyl chloride;[1082] it is used in the manufacture of plastics, synthetic fibers, and numerous other commercial products. There is evidence that inhalation of the substance, either during its production or its use in the manufacture of other materials, may be associated with chronic pulmonary disease. Epidemiologic studies have shown the presence of radiographic abnormalities consistent with pneumoconiosis in 3% to 20% of workers.[1083–1085] Evidence of obstructive pulmonary function was identified in almost half of workers in one investigation. Occasional case reports in humans and experimental studies in animals have also documented a possible association between exposure and the presence of interstitial pneumonitis and fibrosis or the accumulation of interstitial and intra-alveolar macrophages.[1082, 1086–1089] An immunologically-mediated multisystem disorder manifested by Raynaud's phenomenon, acro-osteolysis, thrombocytopenia, portal fibrosis, and hepatic and pulmonary dysfunction has also been ascribed to both polyvinyl chloride and vinyl chloride.[1089a, b, c]

Titanium Dioxide

Titanium dioxide (rutile, anatase) is derived from the ore ilmenite and is used chiefly as a pigment in paints, paper, and other products; as a mordant in dyeing; as a food additive; and as an alloy in some hard metals. Pathologic examination of the lungs of workers who have been in contact with the substance has generally shown alveolar and interstitial accumulation of pigment-laden macrophages but

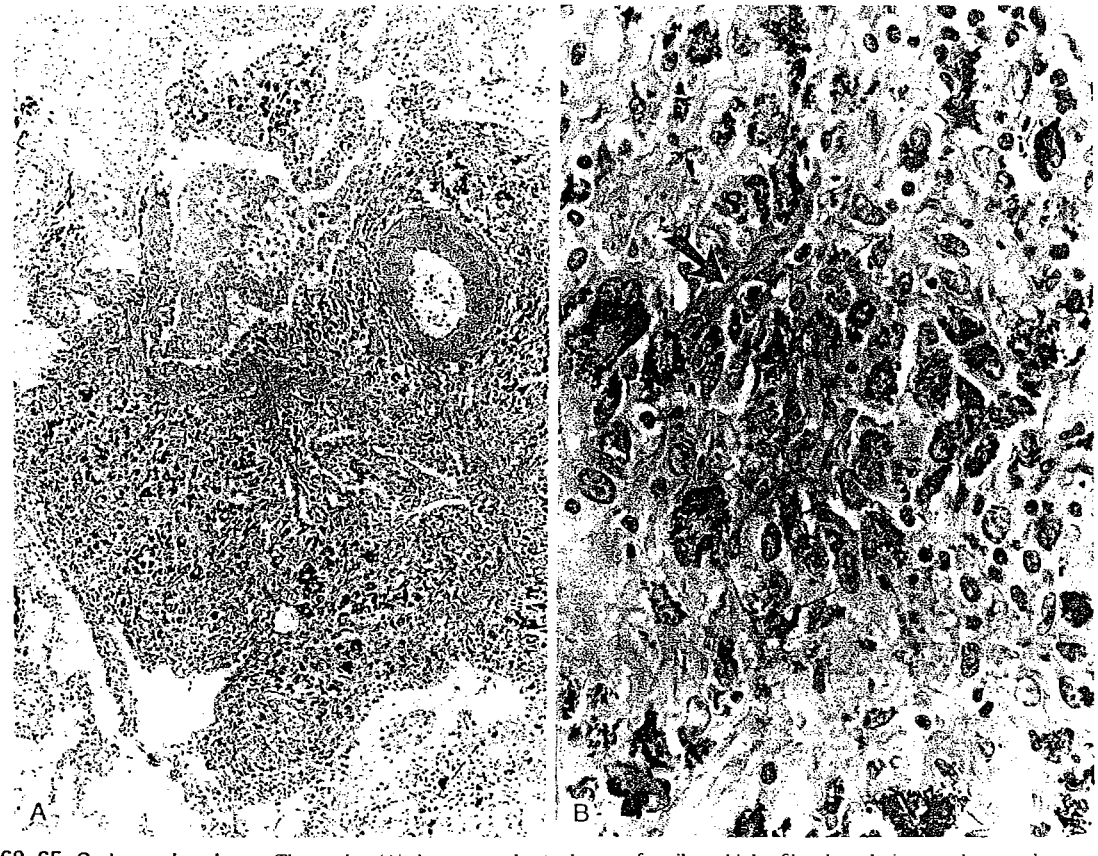

Figure 60–65. Carborundum Lung. The section *(A)* shows a moderate degree of peribronchiolar fibrosis and pigmented macrophage accumulation. Higher magnification *(B)* shows macrophages to contain abundant "anthracotic" pigment and scattered ferruginous bodies *(arrow)* that have a black fibrous core, representing carborundum. (*A*, ×60; *B*, ×250.)

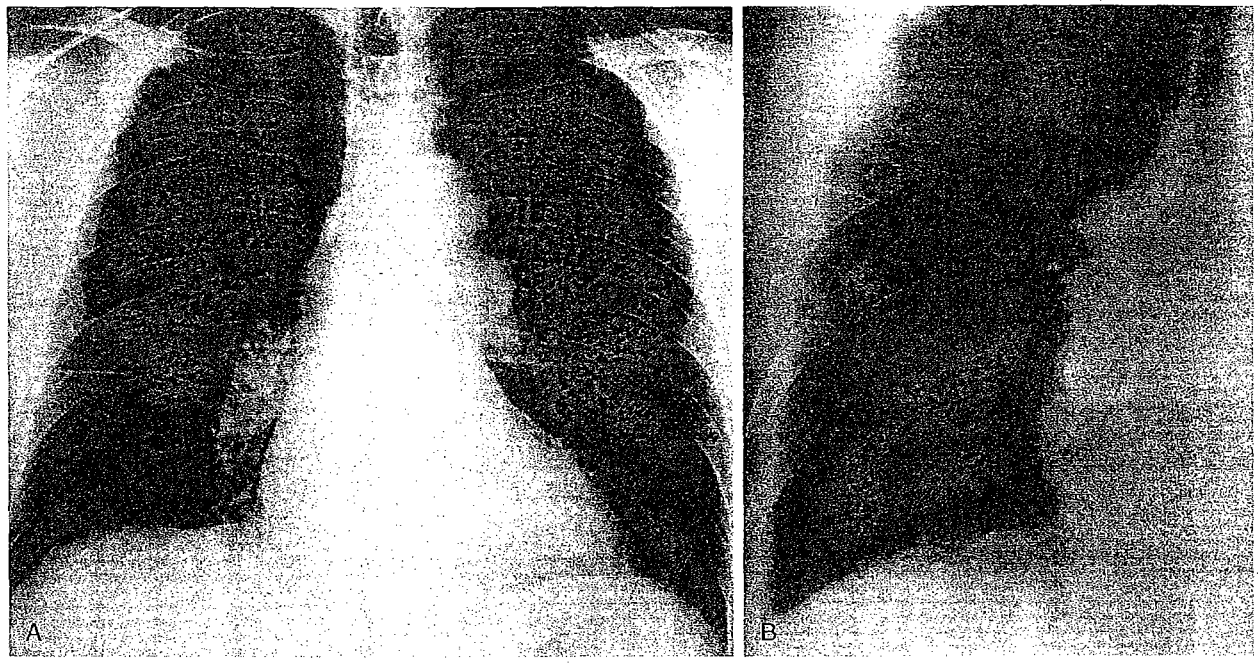

Figure 60–66. Carborundum Lung. A posteroanterior chest radiograph *(A)* demonstrates bilateral hilar lymphadenopathy as well as small nodular and irregular linear opacities involving mainly the lower lung zones. A magnified view of the right lower lung *(B)* better demonstrates the fine reticulonodular pattern and interlobular septal thickening. The patient had been exposed to carborundum for 33 years in a factory manufacturing abrasives. (Courtesy of Dr. Gaston Ostiguy, Maisonneuve-Rosemont Hospital, Montreal.)

no[1090] or minimal[1091] fibrosis. The apparent innocuity of the material has been corroborated by experimental studies in animals.[1091] Despite these observations, clinical and radiographic disease and histologically evident interstitial fibrosis have been documented in some patients,[1093] and the inertness of the substance has been questioned.

Nonnecrotizing granulomatous inflammation was identified in a biopsy specimen from one patient;[1094] because of a positive lymphocyte transformation test on exposure to titanium, the authors considered the possibility of a hypersensitivity reaction similar to that proposed for berylliosis. In macrophages, titanium dioxide appears as small black granules similar to "anthracotic" pigment (Fig. 60–67); however, in contrast to the latter, they are strongly birefringent.[1091, 1092] Radiographic changes considered consistent with pneumoconiosis have been reported in workers involved in pigment production.[1090–1092] A cross-sectional survey of 209 titanium metal production workers showed a reduction in ventilatory capacity and radiographic evidence of pleural plaques and thickening not clearly attributable to asbestos exposure.[1095] In another investigation of 67 workers in a paint factory in Nigeria, almost 50% were found to have pulmonary symptoms (chest pain, cough) and about 40% to have functional evidence of restrictive lung disease.[1080]

Volcanic Dust

Volcanic eruption occurs when magma (liquid rock) is extruded from the depths of the earth to its surface. Although the magma may simply flow over the rim of the volcano onto the adjacent earth (where it is known as lava), violent eruption into the atmosphere can also occur and can produce large amounts of ash (tephra). Depending on the severity and nature of the eruption (e.g., whether it is vertical or at an angle to the earth's surface) and on the composition of the magma itself, significant quantities of potentially harmful ash may be spewed into the atmosphere.

The best-studied volcanic eruption from the point of view of human health occurred at Mount Saint Helens in 1980.[1096] As of 1981, 35 individuals were known to have died directly as a result of the eruption;[1097] among the 25 who underwent autopsy, the majority were considered to have asphyxiated as a result of major airway plugging by mucus and inhaled volcanic ash.[1098] In individuals outside the areas of most severe damage, there was a mild increase in the number of acute respiratory complaints, such as cough, wheezing, and dyspnea, probably secondary to airway irritation.[1096] A considerable increase in emergency department attendance by patients who had asthma and bronchitis was also recorded at local hospitals.[1099]

The long-term consequences, if any, of volcanic ash inhalation are unclear. It has been estimated that free crystalline silicates formed about 3% to 7% of the ash of the Mount Saint Helens eruption,[1096] and it is conceivable that persons who suffered heavy exposure might develop chronic pulmonary disease, presumptively and rather remarkably designated *pneumonoultramicroscopicsilicovolcanoconiosis*![1100] Whether individuals exposed to ash derived from other volcanic sites also have a risk for the development of disease is unclear.

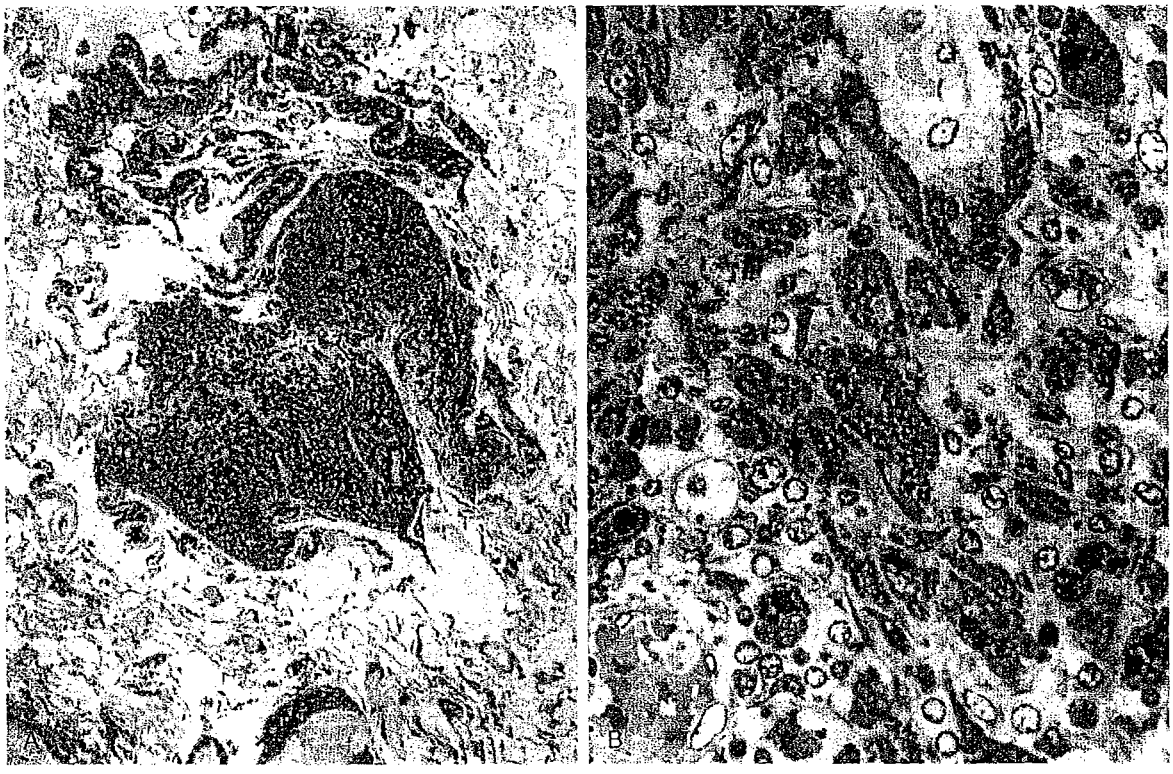

Figure 60–67. Titanium Lung. A section of lung shows patchy, moderately severe interstitial thickening *(A)* caused by an accumulation of numerous macrophages *(B)* containing finely granular black pigment; there is minimal fibrosis. (*A*, ×25; *B*, ×630.) (From Moran CA, Mullick FG, Ishak KG, et al: *Identification of titanium in human tissues.* Hum Pathol 22:450, 1991.)

Synthetic Mineral Fibers

Synthetic mineral fibers are amorphous silicates derived from industrial slag, volcanic rock, ceramic, or glass. Their diameter and length vary considerably, depending on the specific use to which they are put;[1101] for example, fibers used in textiles and as reinforcement in plastics and other materials are mostly between 9 and 25 μm in diameter, whereas those employed in insulation are generally smaller (3 to 6 μm). In contrast to natural silicates, such as asbestos, synthetic fibers break transversely rather than longitudinally when traumatized, resulting in small fragments whose diameter is the same as that of their parents.[1101] Because the potential for causing disease is related to a high length-to-diameter ratio, at least in part,[467, 1101, 1102] this effect may be important in explaining the relative lack of toxicity of these substances.

The bulk of evidence suggests that inhaled synthetic mineral fibers have little, if any, harmful effects on the lungs.[1101] In one autopsy study, no gross or microscopic abnormality was found in the lungs of workers exposed to fiberglass;[1101] in addition, the total number of fibers per gram of dry lung was similar to that of a control group, implying adequate clearance of inhaled particles.[1103] Inhalation of synthetic mineral fibers by rats, hamsters, and monkeys has failed to cause significant fibrosis or neoplasia;[1101, 1104] no alteration of pulmonary structure or inflammatory reaction has been observed except for the presence of alveolar macrophages during the early stages and the development of proteinosis in some animals after 90 days of inhalation.[1105] In addition to these pathologic studies, most epidemiologic investigations, radiographic surveys, and tests of pulmonary function of workers exposed to synthetic mineral fibers have shown no differences from those of appropriate controls.[1101, 1104, 1106, 1107]

Despite the abundant evidence implying lack of pathogenicity of these fibers, the possibility that toxicity might occur in some situations cannot be entirely excluded. It has been suggested, for example, that the results cited previously may simply reflect a relatively low dust exposure.[1107, 1108] In addition, the results of some studies have raised the possibility that the fibers can cause significant tissue damage.[1109] For example, in one investigation of 1,448 fiberglass workers, a statistically significant increase in the number of deaths caused by respiratory disease other than cancer and pneumonia was identified compared with controls.[1110] In another survey of workers involved in the manufacture of refractory ceramic fibers, a type of man-made vitreous fiber, pleural plaques were identified in 5 of 19 workers (26%) who had more than 20 years' exposure, a finding that was not explained by asbestos exposure.[1111] In addition, in a murine model of intense fiberglass exposure, the pulmonary response to fiberglass was found to be similar to that described for crocidolite asbestos.[1112]

The results of several investigations of pulmonary function have also raised questions about the lack of toxicity of synthetic mineral fibers.[1113] For example, in one group of appliance manufacturing workers who had little or no asbes-

tos exposure and who had more than 20 years' exposure to fiberglass, pleural and parenchymal changes as well as alterations in lung function similar to those of asbestos-exposed workers were described in 13%.[1114] However, this study has been criticized for its failure to include control radiographs in the interpretation of the films,[1115] for its failure to adjust for smoking intensity,[1115] and for inadequate consideration of the effects of associated asbestos exposure.[1116] In another study of insulation workers exposed to rock and glass wool, the presence of obstructive lung function and a faster rate of decline in FEV_1 compared to a control group of non–dust-exposed bus drivers were identified.[1117] In a third investigation of workers involved in the manufacture of refractory ceramic fibers, a significant decrease in FVC was found in those individuals who had at least 7 years exposure.[995]

On the basis of the results of these various studies, it seems reasonable to conclude that there is a possible, albeit quantitatively uncertain, risk for the development of pulmonary disease after exposure to man-made mineral fibers.

In contrast to the relative benignity of inhaled synthetic mineral fibers, their instillation directly into the pleural or peritoneal cavities of experimental animals has been shown to be associated with the development of mesothelioma.[1102] Some man-made mineral fibers have been shown to cause hydroxyl radical mediated DNA base modification *in vitro*, possibly explaining the fibers' carcinogenicity.[1118] Despite these observations, no association between mineral fiber inhalation and human mesothelioma has been documented. Some investigators have argued that fibrous glass materials are carcinogenic and that they may be as potent in this respect as asbestos on a fiber-per-fiber basis.[1119] However, when cigarette smoking is taken into account, no increase in the prevalence of pulmonary carcinoma has been found in exposed workers.[1120–1124]

Dust Exposure in Dental Technicians

Although the radiographic abnormalities that sometimes develop in dental technicians have been attributed to SiO_2, it is probable that other agents are involved as well: Air concentration exposure studies and mineralogic analyses of BAL fluid and lung tissue of affected patients have disclosed a variety of substances in addition to silica, including chromium, nickel, aluminum, cobalt, molybdenum, beryllium, acrylic resin, and alginate impression powder.[14, 15, 1125–1128] The prevalence of pulmonary disease in this occupation may be significant; in one study of dental technicians who had more than 30 years' exposure, 22% were found to have radiologic evidence of pneumoconiosis.[1129]

Cement Dust

The results of several studies, some epidemiologic and others single case reports, have implicated cement as a cause of pneumoconiosis.[1130] In a radiographic survey of 195 cement workers, many years' exposure to a high concentration of raw and mixed cement dust was found to be associated with the accentuation of linear markings and ill-defined micronodulation;[1131] however, little or no evidence of radiographic abnormality has been found in other studies.[1131, 1132] Although an increased incidence of carcinoma of the stomach was described in cement workers in one study,[1133] no increase in mortality was observed from respiratory disease. It has been speculated that cement dust may be involved in the pathogenesis of chronic air-flow obstruction in tunnelers using the shotcrete method (in which the tunnel is excavated by shooting a mixture of cement, water, and sand under high pressure).[1134] A case of alveolar proteinosis arising in a cement truck driver has also been reported.[1135] It is possible that these abnormalities may have been caused by the quartz and asbestos that are present in varying amounts in some cement.

Zirconium

Zirconium is a heavy metal used as an alloy in the nuclear industry and in the glazing of ceramic tiles. A single case of pulmonary fibrosis has been reported in association with its use.[1136] Granulomatous interstitial disease imitating sarcoidosis or acute hypersensitivity pneumonitis has also been described in some individuals.[1137–1139] Such effects are unusual, however; in one long-term study of 178 men followed from 1975 to 1988, no evidence of radiographic or functional abnormalities related to the mineral was identified.[1140]

Nylon Flock

Flock is finely cut nylon that is used in upholstery, clothing, and automobiles. An excess incidence of chronic diffuse interstitial lung disease has been described in two North American nylon flock production/flocking plants;[1028, 1141] in one study of 165 workers in a plant in Rhode Island, 7 (4%) were affected. Tissue obtained from transbronchial and wedge lung biopsy specimens has demonstrated nonspecific interstitial pneumonitis or (rarely) bronchiolitis obliterans organizing pneumonia;[1028] nodular lymphoid infiltrates with germinal centers have been seen in most patients, particularly in a peribronchovascular distribution. No granulomatous inflammation has been noted, and the precise cause of the abnormalities has not been identified.

In one investigation of eight patients, four had diffuse reticulonodular opacities, one had patchy consolidation, and three had normal chest radiographs.[1028] HRCT demonstrated bilateral patchy areas of ground-glass attenuation in six patients and peripheral honeycombing in the other two patients;[1028] two patients had focal areas of consolidation and one had diffuse micronodularity associated with the areas of ground-glass attenuation. Symptoms of dry cough and dyspnea occur with a mean latency of 6 years after initial exposure. Improvement has been noted after cessation of exposure and with the use of corticosteroids; however, no workers have recovered completely during the reported follow-up period.[1141]

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